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Intensified conventional insulin treatment and neuropsychological impairment

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Abstract

***Objective*—To assess whether intensified insulin treatment, with an increased frequency of hypoglycaemic episodes, leads to cognitive deterioration.**

***Design*—Prospective randomised trial of intensified conventional treatment and standard treatment.**

***Setting*—Outpatient clinic for patients with insulin dependent diabetes.**

***Subjects*—96 patients with insulin dependent diabetes, high blood glucose concentrations, and non-proliferative retinopathy were randomised to intensified conventional treatment (n=44) or standard treatment (n=52).**

***Main outcome measures*—Glycated haemoglobin concentration (metabolic control); the number of hypoglycaemic episodes reported by patients at each visit; results of computerised neuropsychological tests performed at entry and after five years.**

***Results*—Mean glycated haemoglobin concentration during the study was 7·2% (SE 0·1%) with intensified conventional treatment and 8·7 (0·1%) with standard treatment ($p<0·001$). During five years 34 (77%, 95% confidence interval 53% to 100%) of the patients given intensified treatment and 29 (56%, 36% to 75%) of the others had at least one episode of serious hypoglycaemia ($p<0·05$). The intensified conventional treatment group had a mean of 1·1 episodes of serious hypoglycaemia per patient per year compared with 0·4 episodes in the standard treatment group. Results of the neuropsychological tests were similar in the two groups after five years.**

***Conclusions*—Intensified conventional insulin treatment led to lower blood glucose concentrations and a higher frequency of hypoglycaemic episodes, but patients showed no signs of cognitive deterioration.**

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Introduction

Intensified insulin treatment retards the development of microvascular diabetic complications at the expense of increasing the frequency of serious hypoglycaemic episodes.[1] After three years of intensified conventional treatment the hypoglycaemic episodes had not caused any permanent cortical dysfunction,[2] although earlier studies found that patients with serious hypoglycaemia showed neuropsychological deficits.[3-5] These studies, however, were either non-randomised or cross sectional and retrospective.

TABLE I—*Characteristics of diabetic patients given intensified conventional treatment and standard treatment at entry. Values are means (SE) unless stated otherwise*

Treatment group	No (male/female)	Duration of diabetes (years)	Age (years)	Insulin dose (IU/kg)	Body mass index (kg/m²)
Intensified conventional	44 (22/22)	18·0 (1·0)	29·5 (1·1)	0·73 (0·03)	22·5 (0·3)
Standard	52 (27/25)	16·1 (0·7)	31·6 (1·0)	0·75 (0·03)	22·8 (0·4)

To clarify the long term effects of episodic hypoglycaemia on the brain has been suggested as a major scientific task.[6] We report the results of computerised neuropsychological tests after five years in patients randomised to intensified conventional insulin treatment or standard treatment.

Patients and methods

One hundred and two patients were selected for entry to the study, and after five years 96 patients remained in the study.[1] Five patients had died and one had moved away from Stockholm. The patients initially had insulin dependent diabetes, non-proliferative retinopathy, and unsatisfactory blood glucose control, as already described.[1] They were randomised to intensified conventional treatment (n=48 at entry and 44 after five years) or standard treatment (n=54 at entry and 52 after five years). The treatment regimens have been described.[7] The groups were similar with regard to sex distribution, age, duration of diabetes, insulin dose, body mass index (table I), smoking habits, alcohol consumption, and initial microvascular complications.[1 7]

After three years an effort was made to reduce glycated haemoglobin concentration below 9% in all the patients given regular treatment as nephropathy had been shown to increase steeply with higher concentrations.[8] Two patients receiving standard treatment could not participate in the neuropsychological tests after five years because of reduced visual acuity.

Glycated haemoglobin concentration (normal range 3·9-5·7%) was measured as described previously[1 7] and the numbers of serious hypoglycaemic episodes (during which the patient required help from someone else) and episodes of unconsciousness were carefully recorded and reported by the patients at each office visit.[1 2] The symptoms during hypoglycaemia were recorded at baseline and after five years and were categorised as either predominantly adrenergic or neuroglycopenic.[2 9]

NEUROLOGICAL EXAMINATIONS

Motor and sensory nerve conduction velocities were analysed in the ulnar nerve.[10] Vibration and thermal thresholds were measured on the hand.[11 12] Examinations were performed at entry to the study and after five years on the patient's dominant side and with his or her skin temperature well controlled.

NEUROPSYCHOLOGICAL TESTS

The automated psychological test system measures cerebral dysfunction by a battery of computerised neuropsychological tests.[13-16] It works on an Apple II microcomputer with a custom made keyboard. Each test was performed twice, two days apart to exclude training effects, the first time as an exercise. Only data from the second session were used for analysis. Each

complete test session took 45 minutes. The tests used and the order of use are given below.

Finger tapping test assesses frontal lobe functions such as the motor ability and the ability to switch between the two hemispheres. Its five subsets were (*a*) right index finger tapping, (*b*) left index finger tapping, (*c*) alternate right index and middle finger tapping, (*d*) alternate left index and middle finger tapping, (*e*) alternate tapping of the right and left index fingers. Each test lasted for 12·5 s, and the results are given as the mean number of tappings or alternations a second.

Reaction time test assesses both attention and a combination of cortical functions. Its four subsets were (*a*) simple auditory reaction time, with 10 auditory stimuli of 1000 Hz at around 65 dB; (*b*) simple visual reaction time, with 10 light stimuli (22×22 mm) in the centre of a screen; (*c*) two choice (left-right) visual reaction time, with 10 stimuli to the right and 10 to the left of the screen (80 mm from the centre) in random order; (*d*) two choice visual reaction time with auditory inhibition, as described for (*c*) but in 11 cases the right stimulus was given with an auditory signal, which should cause the subject not to react to the light stimulus. In tasks (*a*) and (*b*) subjects responded with their preferred hand and in tasks (*c*) and (*d*) with the hand on the same side as the visual stimulus.

Necker cube test reflects frontal lobe functions. A cube was presented in the middle of the screen and the subjects looked passively at a central fixation point for 90 seconds. They pressed a button every time the cube changed in perspective. The number of passively perceived perspective reversals were recorded. Patients with unilateral frontal lobe damage show a decrease in the number of reversals; bilateral damage often leads to an increase in reversals.[17]

Trail making test assesses visuomotor coordination, visual search, and eye to hand coordination (frontal lobe functions and switches between the right or left hemispheres and the frontal lobe). It is very sensitive to brain injury.[18] In two sessions numerals (1 to 9) alone were used and in one session both numerals and letters. All the sessions were performed with both the dominant and the non-dominant hand. The numerals and letters were presented on the monitor screen. By moving a cursor controlled by a joystick the patient hit each character in serial order (from 1 to 9 or from 1,A to 9,J). The scores presented are the mean time between hits in seconds.

Digit span test reflects short term memory and concentration and is thus affected by brain damage in many regions.[19] A series of numerals were presented on the screen. In the first part of the test the subject repeated the series forwards—that is, in the given order—and in the second part backwards—that is, in reverse order. The forward task started with three numerals; altogether, 13 series were given. The backward task started with two numbers and consisted of 10 series. If the subject gave a correct answer the next series contained one more number and if an incorrect answer one fewer. For both the forward and the backward series the scores given are the maximum number of numerals and the median of the last three series.

Maze test is complex, reflecting visuospatial skill, motor speed, and strategy. Originally designed as a test of frontal lobe function after surgical procedures, it has since been shown to reflect general intelligence.[20] The subjects constructed a pathway through a maze as fast as possible and passing as many marked nodes as possible. They either were told the number of nodes required for a correct solution (with target information) or were not told (without target information, see table IV). The number of nodes processed per second, check time (time after completing the pathway until pressing a confirm button), motor time (time taken for the most rapid key pressing), and the number of rubbings out were calculated.

Blood glucose concentrations were checked immediately before and after each test. The tests were performed only when blood glucose values were above 4·5 mmol/l. The results from all the tests were compared in the two groups after five years.

STATISTICAL METHODS

Results are given as means and differences between means with 95% confidence intervals[21] or as means (SE). The two groups were compared with Student's *t*

TABLE II—*Mean results from tapping test and mean reaction times after five years in intensified conventional and standard treatment groups*

	Treatment group		Difference between means (95% confidence interval)
	Intensified conventional	Standard	
	Tapping test (taps/s)		
Hand:			
Right	6·8	6·5	0·3 (−0·1 to 0·7)
Left	6·2	6·1	0·1 (−0·1 to 0·3)
Right alternating	3·8	3·9	0·1 (−0·5 to 0·7)
Left alternating	3·4	3·3	0·1 (−0·5 to 0·7)
Left-right alternating	4·1	4·1	0 (−0·6 to 0·6)
	Reaction time (ms)		
Auditory	209	207	2 (−10 to 14)
Visual:			
Alone	241	241	0 (−16 to 16)
Two choices:			
Left	303	309	6 (−10 to 22)
Right	297	295	2 (−15 to 19)
No of errors	1·5	1·7	0·2 (−0·1 to 0·5)
Two choices with auditory inhibition:			
Left	442	440	2 (−40 to 44)
Right	433	441	8 (−12 to 28)
Two choices with random auditory inhibition:			
No of failed inhibitions	1·1	0·9	0·2 (−0·1 to 0·5)

No significant differences between groups.

TABLE III—*Mean results from Necker cube, digit span, and trail making tests after five years in intensified conventional and standard treatment groups*

Test	Treatment group		Difference between means (95% confidence interval)
	Intensified conventional	Standard	
Necker cube (No of reversals)	16	15	1 (−4 to 6)
Digit span (No of numerals remembered):			
Forward:			
Maximum	8·3	8·2	0·1 (−0·4 to 0·6)
Median of last three series	7·3	7·2	0·1 (−0·4 to 0·6)
Maximum	7·8	7·6	0·2 (−0·3 to 0·7)
Median of last three series	6·9	6·7	0·2 (−0·3 to 0·7)
Trail making (time between hits (s)):			
Non-dominant hand:			
Numerals first	2·1	2·0	0·1 (−0·2 to 0·4)
Numerals second	1·9	1·8	0·1 (−0·2 to 0·4)
Numerals and letters	2·3	2·4	0·1 (−0·2 to 0·4)
Dominant hand:			
Numerals first	1·9	2·1	0·2 (−0·1 to 0·5)
Numerals second	1·8	1·9	0·1 (−0·2 to 0·4)
Numerals and letters	2·1	2·4	0·3 (0 to 0·6)

No significant differences between groups.

TABLE IV—*Mean results from maze test after five years in intensified conventional and standard treatment groups*

	Treatment group		Difference between means (95% confidence interval)
	Intensified conventional	Standard	
	With target information		
Process rate (nodes/s)	6·4	6·8	0·4 (−0·8 to 1·6)
Check time (s)	0·56	0·56	0 (−0·08 to 0·08)
Motor time (ms)	364	379	15 (−24 to 59)
Rubbings out (No/maze)	0·7	1·1	0·4 (0 to 0·8)*
	Without target information		
Process rate (nodes/s)	5·8	6·3	0·5 (−0·8 to 1·8)
Check time (s)	0·62	0·62	0 (−0·12 to 0·12)
Motor time (ms)	367	387	20 (−20 to 60)
Rubbings out (No/maze)	0·4	0·5	0·1 (−0·1 to 0·3)

*p<0·05; only significant difference between groups.

test, the Mann-Whitney U test, or a χ^2 test; p values above 0·05 were considered to be not significant.

The study protocol was approved by the ethics committee of the Karolinska Institute, Stockholm. The patients gave their informed consent before participating in the study.

Results

Glycated haemoglobin concentration at entry was 9·5% (0·2%) in the group randomised to intensified conventional treatment and 9·4% (0·2%) in that randomised to standard treatment. During the study (the mean of 14 values) it was 7·2% (0·1%) with intensified treatment and 8·7% (0·1%) with standard treatment ($p<0·001$).

As was previously reported,[1] the patients given intensified treatment had more frequent serious hypoglycaemic episodes than did those given standard treatment. Over five years 34 patients (77%, 95% confidence interval 53% to 100%) receiving intensified treatment and 29 (56%, 36% to 75%) of those receiving standard treatment had at least one episode of serious hypoglycaemia ($p<0·05$).[1] Eighteen (41%, 20% to 61%) of the intensified treatment group and 10 (19%, 0% to 42 %) of the other group were unconscious at least once ($p<0·05$). The mean total number of serious hypoglycaemic episodes was higher in the group given intensified treatment (1·1 *v* 0·4 per patient per year). Of the 32 patients in the intensified conventional treatment group who initially had adrenergic symptoms during hypoglycaemia, 19 changed to having neuroglycopenic symptoms (59%, 42% to 76%). The corresponding figures for the other group were 10 out of 36 patients (28%, 13% to 43%) ($p<0·01$).

Neurological examinations—Conduction velocity in the ulnar motor nerve was slightly higher in the group given intensified conventional treatment after five years (56·4 (0·9) *v* 53·6 (0·7) m/s, $p<0·05$), but none of the patients from either group had a conduction velocity below the lower limit of normal. Conduction velocity in the sensory nerve, the vibration threshold, and the thermal threshold did not differ significantly between the groups.

Neuropsychological tests—There were no significant differences between the groups in the results of the neuropsychological tests (tables II-IV). The only exception was the number of rubbings out in the maze test (table IV), which was slightly lower in the group receiving intensified conventional treatment.

Discussion

Hypoglycaemia is sometimes an obstacle to improving control of blood glucose concentration, and it is more frequent in patients with lower blood glucose concentrations.[22] The frequency of hypoglycaemic episodes was higher in the patients receiving intensified treatment and the increase was seen in patients who had a mean glycated haemoglobin concentration as high as 8·0%[2]—that is, approximately 1·7 times the normal mean. After three years we noted that the increased frequency of serious hypoglycaemic episodes was related to the tendency to change from adrenergic to neuroglycopenic symptoms during hypoglycaemia.[2] Over five years this tendency was more pronounced in the group receiving intensified treatment, which lowered blood glucose concentrations more than did standard treatment. The reason for this change in symptoms, which has also been noted by other investigators,[23] is unknown, but it is not caused by autonomic neuropathy.[2] One of the reasons might be a downward shift of the glucose threshold for hormonal counterregulation.[24]

Episodic hypoglycaemia might cause permanent brain damage.[3-5] In rats neuronal damage occurred only in rats who had isoelectric electroencephalograms during hypoglycaemia.[25] The patients given intensified treatment more often needed help from someone else and also had a higher frequency of hypoglycaemic coma.

We used a battery of neuropsychological tests to analyse brain damage affecting the cerebral hemispheres, especially the frontal lobes. In rats profound hypoglycaemia led to diffuse neuronal damage in the cerebral cortex.[26,27] Our tests were well suited to detect cognitive deterioration secondary to this kind of brain damage in humans. When some of these tests were used during acute hypoglycaemia the results deteriorated with low blood glucose concentrations.[28-30] As the anatomical site of a hypothesised post-hypoglycaemic brain injury cannot be exactly localised in humans, the tests used should cover a broad range of functions. Some of our tests (such as the digit span, trail making, and maze tests) have a high sensitivity for detecting diffuse organic brain damage, especially frontal lobe lesions. The Necker cube test differentiates unilateral from bilateral frontal lobe damage, and the tapping test assesses motor function and coordination between the two hemispheres.

The results from the tapping and the reaction time tests could be influenced by peripheral neuropathy. When neuropathy in the legs and feet was evaluated, deterioration was more pronounced in the group given standard treatment.[1] Conduction velocity in the ulnar motor nerve was slightly higher in the group receiving intensified treatment, but all the patients had normal values. This difference probably did not influence the results of the tapping or reaction time test.

The number of rubbings out in one of the maze tests was lower in the group given intensified treatment. In the absence of other differences this is of no importance and probably a chance finding.

ASSESSING EFFECTS OF HYPOGLYCAEMIA

Six of the patients originally included in the study were not examined after five years because they had either died or moved away. One of the patients who died had experienced frequent hypoglycaemic episodes, but this had been the case before he entered the study.[1,31] Otherwise, the patients who died did not differ much from the survivors with regard to hypoglycaemia, and their exclusion is unlikely to have influenced the results significantly.

Some previous studies have shown poorer cognitive function in patients with more frequent serious hypoglycaemia[3-5] or generally lower blood glucose concentrations.[32] In an often cited work by Bale the blood glucose values before and during the tests were not measured[3] and the test results are therefore impossible to evaluate.

The other studies have also been retrospective and cross sectional. Of special interest is the article by Wredling *et al*, in which blood glucose values were controlled before the test and the automated psychological test system was used.[5] In this study the patients who had experienced serious hypoglycaemia had some signs of neuropsychological defects. Because the study was cross sectional and retrospective, it could not prove a causal relation between hypoglycaemia and brain function. In our study patients were randomised to either intensified or regular treatment. The treatment groups were similar at entry to the study, whereas the groups studied by Wredling *et al* might have differed from each other. Our patients had an increased frequency of hypoglycaemic episodes when they lowered their glycated haemoglobin concentrations but not before this. In the study by Wredling *et al* serious hypoglycaemia appeared "spontaneously"—that is, without intensification of insulin treatment.

The differences observed in neuropsychological test results might represent a trait characteristic of a particular group of patients who have frequent hypoglycaemia without having received intensified insulin treatment. The authors were also well aware of this possibility.[5]

Diabetic patients with near normoglycaemia differ from people without diabetes with regard to substrate utilisation in the brain and regional cerebral blood flow during normoglycaemia.[33] These diabetic patients did not, however, differ from their non-diabetic counterparts in neuropsychological function.[33] The differences observed in blood flow and substrate utilisation are therefore probably not related to any anatomical defects of clinical significance.

Intensified conventional insulin treatment, which retarded the development of microvascular diabetic complications, led to an increased frequency of serious hypoglycaemic episodes, during which patients needed help from someone else and which more often resulted in hypoglycaemic coma. Although these episodes were upsetting for patients and potentially dangerous—for example, if they occurred when driving a car—they did not cause permanent cognitive deficit.

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Training and supervision of obstetric senior house officers

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Recent reports suggest that there are inadequacies in the training of junior hospital doctors.[1-3] We carried out a study at four teaching hospitals and three district general hospitals to examine training of junior hospital doctors in obstetrics.

Subjects, methods, and results

Doctors were questioned on training, communication, and supervision and on the amount of responsibility they were given, and whether they had been involved in incidents and emergencies, and what the outcome was. The doctors were interviewed twice with the same format, once in their first month in an obstetric unit and again five months later. All senior house officers in each unit were interviewed.

At the first interview there were 39 senior house officers in the study, and at the second interview 26.

The study focused on two aspects: use of forceps and cardiotocograms; these were aspects of most concern according to an analysis of cases that had come to litigation.[1] Training in the use of forceps was defined as being shown by a registrar how to use forceps and using them at least once with a registrar in attendance. Training in cardiotocography was defined as formal training such as a tutorial or, at least, a registrar reviewing some cardiotographs and explaining what is or is not an abnormal or equivocal trace.

At the end of six months in an obstetrics unit six (23%) senior house officers had had no training in the use of forceps, although three of them said that they had used them. Of the 20 senior house officers who had been trained, seven (35%) thought that their training had been less than adequate. Half (13) of the senior house officers had had no formal training in interpreting or recognising abnormal or equivocal cardiotocograms, most of whom said that they had learnt what they knew by trial and error, their mistakes being identified and pointed out to them later by midwives and, sometimes,